59. General labourers

Four studies were found which investigated the possible association between lung cancer risk and employment as a general labourer and details of these are given in Table 59. Ten standardized mortality ratios, lying in the range 84-270, were calculated, of which eight were raised. In addition, two proportional mortality ratios, of 106 and 109, were presented, along with two proportional registration ratios, of 115 and 119, and one relative risk estimate of 0.9.

None of the studies gave any information on the possible exposures of the subjects, and no hypotheses were put forward to explain the increase in lung cancer risk observed. Thus, although the evidence presented in the table appears to suggest a slightly higher risk of lung cancer among general labourers, it is not possible to identify any potentially carcinogenic agents which may have been responsible for this increase.

Table 59: Estimates of standardized mortality ratio/relative risk for general labourers

Study	Population	Standardized mortality ratio
OPCS (1978)	E/W male labourers/unskilled workers aged 15-64	109($p<0.01$)[1]
	Aged 65-74	106($p<0.01$)[1]
	Incidence 1966-7	115($p<0.01$)[2]
	1968-9	119($p<0.01$)[2]
Logan (1982)	E/W male labourers - 1931	96
	1951	169
	1961	161
	1971	199
	Married women[3] - 1931	84
	1951	106
	1961	147
	1971	193

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Footnote to Table 58

1 From International Agency for Research on Cancer (1989)

2 90% confidence interval

References

1. Grandjean P and Andersen O (1991) Lung cancer in filling station attendants. Am J Ind Med, 20, 763-768.

2. International Agency for Research on Cancer (1989) Monographs on the evaluation of carcinogenic risks to humans. Volume 46: Diesel and gasoline engine exhausts and some nitroarenes, 41-185. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

60. Glass workers

Only three studies were found which contained data relevant to an investigation of the possible association between lung cancer risk and working in the glass manufacturing industry and details of these are given in Table 60. Two relative risks, of 2.0 and 2.2, were estimated, and one standardized incidence ratio of 128 was also presented.

Glass workers may be exposed to various potentially carcinogenic substances, including arsenic, metals and asbestos [1,3]. However, none of the studies made any attempt to measure the workers' exposures and thus it is difficult to identify the agent or agents responsible for producing the observed increase in lung cancer risk among glass workers. In any case, from the evidence presented in the table it can be seen that any increase in risk, if it does exist, is not likely to be particularly large.

Table 60: Estimates of relative risk/standardized incidence ratio for glass workers

Study	Population	Relative risk (95% limits)
Milne et al (1983)	US glass manufacturing workers	2.2($p<0.05$)
Wingren and Axelson (1985)[1]	Swedish glass blowers	2.00
Sankila et al (1990)	Finnish glass factory workers	128(99-162)

* Standardized incidence ratio

1 From International Agency for Research on Cancer (1987)

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Table 59 continued

Milne et al (1983)	US male general labourers	0.9*
OPCS (1986)	British male labourers	246(p<0.01)
	Married women[3]	270(p<0.01)

E/W - English/Welsh

* Relative risk

1 Proportional mortality ratio

2 Proportional registration ratio

3 According to husband's occupation

References

1. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London.

2. Milne KL, Sandler DP, Everson RB and Brown SM (1983) Lung cancer and occupation in Alameda County: A death certificate case-control study. Am J Ind Med, 4, 565-575.

3. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

4. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

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61. Gold mining

Details of the five studies found which presented data relevant to the investigation of a possible association between lung cancer and gold mining are given in Table 61. Four relative risks were estimated, ranging from 1.4-7.9, along with one standardized mortality ratio of 100. Finally, one study calculated an annual incidence rate of 205.6/100 000, compared to the general male population.

Gold miners are thought to have potential exposure to several substances which may be carcinogenic, and those which have been discussed include arsenic, asbestos, chromium, nickel, radon and silica [1-5]. However, only two of the studies [1,2] attempted to make any objective measurements of workers' exposures and, with the exception of asbestos [2], all of the above-named substances were found only in very low concentrations, below levels which are thought to pose any risk. Thus, the possible role of any individual exposure is very hard to ascertain.

Although the data presented in the table is consistent with an increase in lung cancer risk among gold miners, the lack of information on exposures such workers are subjected to makes it very difficult to identify the agent or agents responsible. One study which found high levels of asbestos but negligible levels of arsenic, chromium, nickel and radon felt that this was sufficient evidence for the excess of respiratory disease observed to be attributed to asbestos exposure [2]. However, no data was available from the other studies to back this up.

Table 61: Estimates of relative risk/standardized mortality ratio for gold miners

Study	Population	Relative risk (95% limits)
Osburn (1957)[1]	Rhodesian gold miners	205.6[2]
Gillam et al (1976)	South Dakota gold miners	2.70($p<0.05$)[3]
Armstrong et al (1979)	Australian gold miners	1.4($p<0.01$)[3]
Katsnelson and Mokronosova (1979)[4]	Russian gold miners - underground	7.9($p<0.001$)
	Surface	1.6
Brown et al (1986)[5]	US gold miners	100*

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 100-106. IARC, Lyon.

2. Milne KL, Sandler DP, Everson RB and Brown SM (1983) Lung cancer and occupation in Alameda County: A death certificate case-control study. Am J Ind Med, 4, 565-575.

3. Sankila R, Karjalainen S, Pukkala E et al (1990) Cancer risk among glass factory workers: An excess of lung cancer? Br J Ind Med, 47, 815-818.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

62. Haematite and iron oxide

Table 62 gives details of the studies which attempted to relate the risk of lung cancer to exposure to haematite and iron oxide. Ten studies calculated standardized mortality ratios, ranging from 100-176, of which half were raised, while another study reported a raised standardized incidence ratio but did not give details of the results. One study observed a lung cancer incidence 10.7 times higher among iron ore miners than among non-miners, while another reported bronchial cancer to be 2.29 times higher in such workers. Three other studies observed lung cancer incidence of 9.4% in haematite miners compared to 2.0% in controls, bronchogenic carcinoma incidence of 3.3% in iron ore miners compared to 1.5% in controls, and a lung cancer mortality rate of 6.6 per 10,000 compared to the national rate of 4 per 10,000. A further three studies reported that lung cancer mortality was higher in the population of interest than in the controls, but did not give any further details. A total of seven lung or bronchial cancers were observed in the remaining three studies.

No information was available from the studies on the level of exposure to haematite and iron oxide, as none appeared to have attempted to quantify exposure in any way. Thus, it is obviously possible that inaccuracies could have been introduced into the results. Furthermore, the study subjects came from a range of occupations, some of which entail exposure to other substances which may themselves be potentially carcinogenic. For example, miners may be exposed to silica and radon, while welding involves exposure to many metals, particularly zinc. Boiler scalers may be exposed to both silica and soot. In the absence of exposure data it is not possible to determine which, if any, of these substances subjects may have come into contact with, or the potential carcinogenicity of any one material.

Therefore, IARC appear to be justified in their classification of the evidence for the carcinogenicity of haematite and iron oxide to humans as "inadequate". However, the evidence for underground haematite mining with exposure to radon was considered "sufficient" [4].

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Footnote to Table 61

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1980)

2 Incidence rate per 100 000 per year

3 Tumours of respiratory system

4 From Goldsmith et al (1982)

5 From International Agency for Research on Cancer (1987)

References

1. Armstrong BK, McNulty JC, Levitt LJ, Williams KA and Hobbs MST (1979) Mortality in gold and coal miners in Western Australia with special reference to lung cancer. Br J Ind Med, 36, 199-205.

2. Gillam JD, Dement JM, Lemen RA et al (1979) Mortality patterns among hard rock gold miners exposed to an asbestiform mineral. Ann N Y Acad Sci, 271, 336-344.

3. Goldsmith DF, Guidotti TL and Johnston DR (1982) Does occupational exposure to silica cause lung cancer? Am J Ind Med, 3, 423-440.

4. International Agency for Research on Cancer (1980) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 23: Some metals and metallic compounds, 39-142. IARC, Lyon.

5. International Agency for Research on Cancer (1987) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 42: Silica and some silicates, 39-144. IARC, Lyon.

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Table 62: Estimates of standardized mortality ratio for exposure to haematite and iron oxide

Study	Population	Standardized mortality ratio
Stewart and Faulds (1934)[1]	Haematite miners	1 bronchial cancer observed
Vorwald and Karr (1937,1938)[1]	Haematite miners	3 lung cancers observed
Turner and Grace (1938)[1]	UK foundry workers/ metal grinders	Lung cancer mortality higher than in other occupations
McLaughlin et al (1945)[1]	Silver finishers	100
Barrie and Harding (1947)[1]	Silver finishers	100
Kennaway and Kennaway (1947)[1]	UK metal grinders	176
Harding (1948)[1]	Silver finishers	100
McLaughlin et al (1950)[1]/ McLaughlin and Harding (1956)[1]	UK iron/steel foundry workers	>100[2]
Harding and Massie (1951)[1]	Boiler scalers	3 lung cancers observed
Wynder and Graham (1951)[1]	Welders/hot metal workers	Lung cancer cases more often hot metal workers than other diseases
Doll (1953)[1]	Welders/hot metal workers	100
Breslow et al (1954)[1]	Welders/hot metal workers	Lung cancer cases more often hot metal workers than other diseases
Faulds and Stewart (1956)	UK haematite miners	Lung cancer incidence 9.4% vs. 2.0% controls
Podhrazsky (1957)[1]	Czech steel plant workers	Mortality rate 6.6[3] vs. 4 nationally
Braun et al (1960)[1]	French iron-ore miners	Bronchogenic carcinoma incidence 3.3% vs. 1.5% in controls (p=0.01)

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Table 62 continued

Monlibert and Roubille (1960)[1]	French iron-ore miners	Bronchial cancer incidence 2.29 times higher than in controls
Gurevich (1967)[1]	USSR iron-ore miners	Lung cancer mortality 10.7 times higher than among non-miners
Boyd et al (1970)	UK haematite miners	168[4]
Anthoine et al (1979)[5]	French iron ore miners	>100
Axelson and Sjoberg (1979)[5]	Iron foundry workers	100
Tola et al (1979)[5]	Iron foundry workers	>100
Lawler et al (1985)[5]	US iron ore miners	100

1 From International Agency for Research on Cancer (1972)

2 Standardized incidence ratio

3 Per 10,000

4 Estimated from data given

5 From International Agency for Research on Cancer (1987)

References

1. Boyd JT, Doll R, Faulds JS and Leiper J (1970) Cancer of the lung in iron ore (haematite) miners. Br J Ind Med, 27, 97-105.

2. Faulds JS and Stewart MJ (1956) Carcinoma of the lung in haematite miners. J Path Bacteriol, 72, 353-366.

3. International Agency for Research on Cancer (1972) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 1, 29-39. IARC, Lyon.

4. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 216-219. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

63. Hairiness of 2nd phalanx

Table 63 gives details of the only study found which attempted to relate lung cancer risk to hairiness of the 2nd phalanx. It was reported that 3.1% of cancer cases had a hairy 2nd phalanx compared to only 2.1% of controls.

The authors themselves stated that multiple comparisons result in some apparent association due to chance alone, and that the observed relationship between lung cancer risk and hairiness of the 2nd phalanx may be a case in point.

Table 63: Observations on hairiness of 2nd phalanx

Study	Population	Observations
Denoix et al (1958)	French men	3.1% bronchial cancer cases had hairy 2nd phalanx compared to 2.1% controls

* Standardized mortality ratio

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

64. Hashimoto's thyroiditis

Table 64 gives details of the only study found which presented data relevant to an investigation of the possible association between lung cancer risk and Hashimoto's thyroiditis. A standardized mortality ratio of 209 was calculated.

With so little information available it is not possible to evaluate the relationship between lung cancer and Hashimoto's thyroiditis.

Table 64: Estimate of standardized mortality ratio for Hashimoto's thyroiditis

Study	Population	Standardized mortality ratio
Yamashita et al (1979)	Japanese women	209($p<0.05$)[1]

1 Estimated from data given

References

1. Yamashita N, Maruchi N and Mori W (1979) Hashimoto's thyroiditis: A possible risk factor for lung cancer among Japanese women. Cancer Letters, 7, 9-13.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

66. History of lung disease

Details of the studies which investigated the possible association between lung cancer risk and a history of previous lung disease are given in Table 66. Relative risks ranging between 0.76-29.82 were estimated, with 55 out of 62 being above 1.00. Two standardized proportionate mortality ratios, both of 1.50, were also estimated. In addition, one study reported a positive association but did not give any detailed results, while two others stated that tuberculosis patients and pneumonia patients were 20 and 100 times more likely to develop lung cancer than the general population respectively, and one study reported that 10.0% of male and 8.2% of female lung cancer cases had a history of tuberculosis infection. Finally, one study reported finding a correlation between lung cancer mortality in adulthood and tuberculosis mortality in the period corresponding to the childhood of the lung cancer cases.

Most of the studies gathered information on history of lung disease from registers or medical records, or followed-up groups of patients with lung diseases, but several (Alavanja, Campbell, Campbell and Hughes, Wu, Wynder, Wynder and Fairchild) appeared to have based their data on information elicited at interviews with the subjects or their next-of-kin. It is obvious that this could have led to inaccuracies due to mistakes in recalling disease status, particularly in those studies which collected health data from a surrogate respondent. Additionally, no details were given at all on how the data on tuberculosis infection were collected in the study by Zheng.

Although results for several diseases were presented in the table, apart from tuberculosis, and to a lesser extent pneumonia, very few studies reported on each disease. Thus, with the exception of tuberculosis, an unequivocal relationship was not seen for any of the diseases considered. The data presented in the table suggest a positive association between lung cancer risk and a history of tuberculosis infection, but on the whole the estimates of relative risk are not particularly large, and therefore the relationship does not appear to be very strong. Furthermore, a recent review of lung cancer and tuberculosis stated that although patients with active pulmonary tuberculosis had a higher than normal risk of dying from lung cancer, there was "little biological evidence of TB lesions themselves, or TB bacilli per se,

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65. Hexachlorocyclohexanes

Table 65 gives details of the only study found which investigated the possible association between lung cancer risk and exposure to hexachlorocyclohexanes (HCH). A standardized mortality ratio of 180 was calculated.

However, it should be noted that the workers in this study received their exposure through the application of various pesticides, and thus exposure could be to compounds other than HCH, or in addition to HCH. Therefore, IARC felt that it was not possible to evaluate the carcinogenicity of HCH to humans on the basis of this study [1] and classified the evidence as "inadequate" [2].

Table 65: Estimate of standardized mortality ratio for exposure to hexachlorocyclohexanes

Study	Population	Standardized mortality ratio (95% limits)
Barthel (1976)[1]	Agricultural workers	180(140-240)

1 From International Agency for Research on Cancer (1987)

References

1. International Agency for Research on Cancer (1979) Monographs on the evaluation of carcinogenic risk of chemicals to humans. Volume 20: Some halogenated hydrocarbons, 195-223. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 220-222. IARC, Lyon.

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having major roles to play in the carcinogenesis of lung cancer at the moment" [2].

Table 66: Estimates of relative risk for history of lung disease

Study	Population	Dis-ease	Relative risk (95% limits)
Wynder et al (1956)[1]	Women	PNEU	1.00
		TB	1.00
Campbell and Hughes (1960)[1]	Not stated	TB	Lung cancer occurred 20 times more frequently in TB patients than general population
Campbell (1961)[1]	Not stated	TB	Positive association
Steinitz (1965)[2]	Israeli residents	TB	10.0% male and 8.2% female lung cancer cases had history of TB
Wynder and Fairchild (1966)	US men	ANY	4.28(2.79-6.56)[3]
		ASTH	3.93(1.18-13.1)[3]
		EMPH	0.93(0.09-9.24)[3]
		INJ	0.76(0.20-2.88)[3]
		PLEU	2.49(1.29-4.80)[3]
		PNEU	1.70(0.99-2.93)[3]
		TB	0.84(0.22-3.21)[3]
Simecek and Simeckova (1967)[2]	Czech residents	TB	4.2
Aoki et al (1969)[2]	US white men	TB	6.7(4.0-9.6)
	Non-white men	TB	8.6(4.0-13.7)
	US white men active	TB	10.5
	Non-white men "	TB	20.6
	White men, inactive	TB	4.5
	Non-white men "	TB	2.3
	US residents, 1962-3	TB	3.6(1.9-5.3)
	1963-4	TB	1.7(0.4-3.0)
	Japanese men	TB	2.75
	Women	TB	5.48

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Table 66 continued

Aoki et al (1969)[2]/ Ipsen (1967)[2]	US men	TB	3.1
Campbell and Guilfoyle (1970)[2]	Australian men	TB	2.06
Kreus et al (1970)[2]	Finnish residents	TB	2.2
Aoki and Ohtani (1971)[2]	Japanese men, 1964-68	TB	1.41[4]
			1.23[5]
	Women	TB	1.33[4]
			1.54[5]
	Men, 1974-82	TB	4.21[4]
			2.13[6]
	Women,	TB	3.87[4]
			2.16[6]
Watanabe and Kurashima (1977)[2]	Japanese residents	TB	3.56
	Japanese residents	TB	6.93
Clemmensen and Hjalgrim-Nelson (1979)[2]	Danish men, INH+	TB	3.36
	INH-	TB	2.58
	Women, INH+	TB	4.55
Howe et al (1979)[2]	Canadian men	TB	1.50(1.21-1.47)[7]
	Women	TB	1.50(1.11-1.98)[7]
Hongo et al (1981)[2]	Japanese men	TB	4.88
	Women	TB	9.69
Komatsu et al (1981)[2]	Japanese men	TB	2.58
	Women	TB	19.6
Mercer (1981)	English residents	TB	Correlation between lung cancer mortality in adulthood and TB mortality in time period corresponding to childhood
Hinds et al (1982)	Hawaiian women	TB	1.0(0.2-5.5)
	Japanese women	TB	2.0(0.4-10.3)
	Chinese women	TB	2.1(0.3-16.1)
Aoki (1985)[2]	Japanese men	TB	12.0
	Women	TB	5.0

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Table 66 continued

Takatorige et al (1985)[2]	Japanese residents, -1 year follow-up	TB	29.82
	1-2 years	TB	6.36
	2-3 years	TB	5.00
	3-4 years	TB	4.35
	4-5 years	TB	1.27
	5+ years	TB	1.27
Zheng et al (1987)[2]	Chinese residents	TB	1.5(1.2-1.8)
Wu et al (1988)	Not stated	TB	10.0(1.1-90.1)
Sakurai et al (1989)[2]	Japanese women	TB	6.4
Gao et al (1991)[2]	Chinese men	TB	1.72(1.11-2.53)
	Women	TB	2.79(1.79-4.14)
Alavanja et al (1992)	US female residents	ANY	1.2(1.0-1.5)
		ASTH	1.3(0.8-2.1)
		EMPH	2.6(1.5-4.7)
		PLEU	0.9(0.7-1.3)
		PNEU	1.2(1.0-1.6)
		TB	2.0(1.0-4.1)
Ohtsuka et al (19??)	Japanese men	PNEU	Pneumonia sufferers 100 times more likely to develop lung cancer than general population

ANY = any previous lung disease; ASTH = asthma; EMPH = emphysema; INH = Isoniazid treatment; INJ = lung injury; PLEU = pleurisy; PNEU = pneumonia; TB = tuberculosis

1 From Roe and Walters (1965)

2 From Aoki (1993)

3 Estimated from data given

4 Cardiovascular disease controls

5 Gastric cancer controls

6 Ischaemic heart disease controls

7 Standardized proportionate mortality ratio

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Alavanja MCR, Brownson RC, Boice JD and Hock E (1992) Preexisting lung disease and lung cancer among nonsmoking women. Am J Epidemiol, 136, 623-632.

2. Aoki K (1993) Excess incidence of lung cancer among pulmonary tuberculosis patients. Jpn J Clin Oncol, 23, 205-220.

3. Hinds MW, Cohen HI and Kolonel LN (1982) Tuberculosis and lung cancer risk in nonsmoking women. Am Rev Resp Dis, 125, 776-778.

4. Mercer AJ (1981) Risk of dying from tuberculosis or cancer: Further aspects of a possible association. Int J Epidemiol, 10, 377-380.

5. Ohtsuka Y, Tanimura K, Munakata M et al (19??) Idiopathic interstitial pneumonia(IIP) as a risk factor for a lung cancer - a prospective study. Incomplete reference.

6. Roe FJC and Walters MA (1965) Some unsolved problems in lung cancer etiology. Progr Exp Tumor Res, 6, 126-227.

7. Wu AH, Yu MC, Thomas DC, Pike MC and Henderson BE (1988) Cancer Res, 48, 7279-7284.

8. Wynder EL and Fairchild EP (1966) The role of a history of persistent cough in the epidemiology of lung cancer. Am Rev Resp Dis, 94, 709-720.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

67. Hormone therapy in women

Details of the only study found which presented data relevant to an investigation of lung cancer risk in women in relation to having received hormone therapy are given in Table 67. A relative risk of 1.26 was estimated.

With only one study reporting it is not possible to properly evaluate the relationship between lung cancer risk and hormone therapy in women.

Table 67: Estimate of relative risk for hormone therapy in women

Study	Population	Relative risk (95% limits)
Adami et al (1989)	Swedish women	1.26(0.92-1.68)

References

1. Adami H-O, Persson I, Hoover R, Schairer C and Bergkvist L (1989) Risk of cancer in women receiving hormone replacement therapy. Int J Cancer, 44, 833-839.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

68. Hydrazine and derivatives

The five studies found which attempted to relate the risk of lung cancer to exposure to hydrazine and its derivatives are detailed in Table 68. One study looked at exposure to hydrazine itself, while the other four considered isonicotinic acid hydrazide (INH). Seven relative risks were estimated overall, of between 1.10 and 4.6.

None of the studies gave any information on the level of exposure to hydrazine or INH, simply stating whether or not exposure had taken place. In fact, in the study by Hammond, it was stated that only 10% of one of the groups of patients had been exposed to INH. Thus, a raised relative risk was observed among a group of patients who were predominately not exposed to INH.

Not surprisingly, IARC felt there was "inadequate" evidence for the carcinogenicity to humans of both hydrazine and INH [2].

Table 68: Estimates of relative risk for exposure to hydrazine and its derivatives

Study	Population	Exposure	Relative risk (95% limits)
Hammond et al (1967)[1]	TB patients	INH	1.23(p>0.05)[2]
	TB patients	INH	1.59(p>0.05)[2]
Campbell and Guilfoyle (1970)[1]	Male TB patients	INH	1.10(p>0.05)[2]
Stott et al (1976)[3]	TB patients	INH	1.6(1.2-2.1)
Clemmensen and Hjalgrim-Jensen (1979)[3]	Male TB patients	INH	3.4
	Female TB patients	INH	4.6
Wald et al (1984)[3]	Male factory workers	HYD	1.2(0.2-4.5)

HYD = hydrazine; INH = isonicotinic acid hydrazide

1 From International Agency for Research on Cancer (1974)

2 Estimated from data given

3 From International Agency for Research on Cancer (1987)

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. International Agency for Research on Cancer (1976) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 4: Some aromatic amines, hydrazine and related substances, N-nitroso compounds and miscellaneous alkylating agents, 159-172. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 223-224 and 227-228. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

69. Hypercalcaemia

Table 69 gives details of the only two studies found which attempted to relate the risk of lung cancer to the presence of hypercalcaemia. A total of 6 lung cancers were observed in association with hypercalcaemia.

With so few studies reporting, and in the absence of any estimates of risk, it is not possible to properly evaluate the relationship between lung cancer and hypercalcaemia.

Table 69: Observations for hypercalcaemia

Study	Population	Observations
Connor et al (1956)	US men	2 cases reported in which hypercalcaemia disappeared after removal of lung tumour
Tashjian (1963)	US residents	4 cases of lung cancer associated with hypercalcaemia

References

1. Connor TB, Thomas WC and Howard JE (1956) The etiology of hypercalcemia associated with lung carcinoma. Am Soc Clin Invest, 35, 697-698.

2. Tashjian AH (1963) Case records of the Massachusetts General Hospital. N Engl J Med, 269, 801-813.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

70. Hypertrophic pulmonary osteoarthropathy

Only two studies were found which gave information relevant to an investigation of the possible association between lung cancer risk and hypertrophic pulmonary osteoarthropathy (HOA), and details of these are presented in Table 70. One study reported that 3-4% of lung cancer cases were also suffering from HOA, while the other noted that 13 of the 14 cases of lung cancer associated with HOA arose from peripherally located tumours, rather than centrally located ones.

Various explanations of the association between the two conditions were put forward, but with so few studies reporting it is not possible to evaluate the relationship between lung cancer and HOA.

Table 70: Observations for hypertrophic pulmonary osteoarthropathy

Study	Population	Observations
Ray and Fisher (1953)	US residents	13 out of 14 cases of HOA associated with lung cancer arose from peripherally located tumours
Coury (1960)	French residents	3-4% lung cancer cases also have HOA

References

1. Coury C (1960) Hippocratic fingers and hypertrophic osteoarthropathy: A study of 350 cases. Br J Dis Chest, 54, 202-209.

2. Ray ES and Fisher HP (1953) Hypertrophic osteoarthropathy in pulmonary malignancies. Ann Intern Med, 38, 239-246.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

71. Iron/steel founding

Table 71 details the numerous studies found which gave some data on the risk of lung cancer in those employed in iron and steel founding and allied occupations. Standardized mortality ratios (SMR) in the range 129-233 were presented by those studies which concentrated on mortality statistics. Three studies gave proportional mortality ratios (PMR), of 123, 170 and 186. The cohort studies showed a similar pattern, with the 25 SMRs presented lying in the range 0-714, with all but one being raised. Two PMRs, of 144 and 147, were also given. Additionally, seven studies produced estimates of relative risk of between 0.96 and 1.39, five of which were above 1.00. Finally, three case-control studies presented eight relative risks of between 1.20 and 7.10. Detailed results were not available from the fourth case-control study, which simply stated that cases were more often floor moulders, coremakers, casters and fettlers than controls.

Iron and steel workers may be exposed to a variety of potentially hazardous substances, including airborne crystalline silica from quartz sand used in moulding and coremaking, metallic fumes present during melting, pouring, welding and flame-cutting, metal dusts associated with abrasive grinding operations, carbon monoxide emitted from the cupola and casting operations, phenol, formaldehyde, furfuryl alcohol, isocyanates and amines used as ingredients of organic binders in mould and core sands, and pyrolysis products, such as polynuclear aromatic compounds, formed as a result of the contact between molten metal and carbonaceous materials during pouring [2]. No information was available from any of the studies as to the actual exposures of the workers, and therefore it is not possible to separate out the effects of any one substance, or even to say which substances the subjects were exposed to.

Despite this drawback, the consistent excess of lung cancer deaths seen in iron and steel foundry workers suggests that certain occupational exposures may be risk factors, and this is reflected in the ratings given by IARC, which stated that such exposures were "probably" carcinogenic [2], although it was later felt that the evidence for carcinogenicity to humans was "sufficient" [3].

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Table 71: Estimates of relative risk/standardized mortality ratio for iron/steel founding

Study	Population	Relative risk (95% limits)
Mortality statistics:		
Turner and Grace (1938)[1]	Foundry/furnace workers, smiths	233*[2]
Swantson (1950)[1]	Furnace men, rollers and assistants	160*
	Metal moulders and diecasters	193*
	Iron foundry furnacemen and labourers	188*
Morrison (1957)[1]	Scottish moulders	162*[2]
	Foundry workers	161*[2]
Enterline and McKiever (1963)[1]	US metal moulders	227*
		170[3]
Adelstein (1972)[1]	E/W furnace/forge/foundry rolling mill workers	123($p < 0.05$)[3]
Milham (1976)[1]	US metal moulders	135*[2]
OPCS (1978)[1]	E/W Moulders	184*($p < 0.05$)
	Fettlers	129*
	Metal furnacemen	155*
Petersen and Milham (1980)[1]	US metal moulders	186[3]
Logan (1982)[1]	E/W Furnace/forge/foundry rolling mill workers - 1961	140*
	1971	155*
Cohort studies:		
Lerer et al (1974)[1]	US foundrymen	1.11
Koskela et al (1976)[1]	Finnish iron/steel/non-ferrous foundry workers	151*
	Iron foundry workers	270*($p < 0.05$)
	Steel foundry workers	0*
	Non-ferrous foundry workers	143*[4]
	Moulders, coremakers	231*[4]
	Casters, furnacemen	238*[4]
	Fettlers	111*[4]
	Labourers	139*[4]

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Table 71 continued

Gibson et al (1977)[1]	Canadian steel casters	250*(p < 0.05)
	Crane operators	714*(p < 0.05)
	Finishers	314*
	Moulding	255*
	Coremaking	208*
Breslin (1979)[1]	US foundrymen - cohort A	1.34 ✓
	Cohort B	0.96
	Cohort C	1.23
Decoufle and Wood (1979)[1]	US grey iron foundrymen working > 5 years	128*[4]
Egan et al (1979)[1]	US foundry workers	147(p < 0.05)[3]
Tola et al (1979)[1]	Finnish iron foundrymen	144(p < 0.05)[3]
Egan-Baum et al (1981)[1]	US white foundry workers	144*(p < 0.05)
	Black workers	176*(p < 0.05)
Redmond et al (1981)[1]	US foundry workers - cohort A	1.39 ✓
	Cohort B	0.99
	Cohort C	1.30
Fletcher and Ades (1984)[1]	E/W steel foundrymen	137*[4]
	Foundry workers per se	142*(p < 0.05)
	Fettling shop workers	173*(p < 0.05)
	Other workers	110*
	Furnace repairmen	203*(p < 0.05)
	Labourers	139*
	Fettlers	195*(p < 0.05)
	Heat treatment workers	356*(p < 0.05)
	Maintenance fitter's mate	225*(p < 0.05)
Case-control studies:		
Tola et al (1979)[1]	Iron foundry workers	Cases more often floor moulders, core makers, casters and fettlers than controls.
Egen-Baum et al (1981)[1]	Iron foundry workers	2.36(1.01-5.53) ✓
Neuberger et al (1982)[1]	Dust-exposed workers[5]	1.29(1.01-1.65)

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 71 continued

Blot et al (1983)	Crane operators, chainmen	1.40(0.60-3.50)
	Furnace workers	2.60(1.20-5.80)
	Machinists	1.60(0.70-4.00)
	Coke worker	1.20(0.20-6.90)
	Foundry worker, coremaker	7.10(1.20-42.3)
	General maintenance workers	1.80(0.80-4.30)

E/W = England and Wales

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1984)

2 Cancers of respiratory system

3 Proportional mortality ratio

4 Estimated from data given

5 Results for iron foundry workers, who formed the largest proportion, were similar to those shown for whole group

References

1. Blot WJ, Brown LM, Pottern LM, Stone BJ and Fraumeni JF (1983) Lung cancer among long-term steel workers. Am J Epidemiol, 117, 706-16.

2. International Agency for Research on Cancer (1984) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 34: Polynuclear aromatic compounds, part 3, industrial exposures in aluminium production, coal gasification, coke production, and iron and steel founding, 133-192. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of the carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 224-225. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

72. Keeping pet birds

Three studies investigated the possible association between lung cancer risk and keeping pet birds, and details of these are given in Table 72. Relative risks ranging from 1.29-6.7 were estimated.

The data presented in the table suggest an association between lung cancer risk and the keeping of pet birds, but with only three studies reporting such a relationship cannot yet be considered proven.

Table 72: Estimates of relative risk for keeping pet birds

Study	Population	Relative risk (95% limits)
Holst et al (1988)/Holst (1988)	Dutch residents	6.7(2.2-20)
Gardiner et al (1992)	Scottish residents	1.29(0.79-2.12)
Kohlmeier et al (1992)	German residents	2.14(1.35-3.40)

* Standardized mortality ratio

References

1. Gardiner AJS, Forey BA and Lee PN (1992) Avian exposure and bronchogenic carcinoma. Br Med J, 305, 989-992.

2. Holst PAJ (1988) Bird keeping as a source of lung cancer and other human disease. A need for higher hygienic standards. Springer-Verlag, Heidelberg.

3. Holst PAJ, Kromhout D and Brand R (1988) Pet birds as an independent risk for lung cancer. Br Med J, 297, 1319-1321.

4. Kohlmeier L et al (1992) Pet birds as an independent risk factor for lung cancer: Case-control study. Br Med J, 305, 986-989.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

73. Lead and lead compounds

Details of studies which gave information relevant to an investigation of the possible association between lung cancer risk and exposure to lead and lead compounds are given in Table 73. Nine standardized mortality ratios, ranging from 110-300, were calculated along with one standardized incidence ratio of 128. In addition, one study reported that levels of lead found in the lungs of patients who had died of lung cancer were similar to those in patients who had died of other causes.

Only two of the studies appeared to have taken objective measurements of the workers' exposure to lead, by analyzing levels in the lungs (Jecklin) or blood and urine (Cooper and Gaffey). No information on exposure was available from the other studies, which could therefore contain inaccuracies. Additionally, workers in at least three of the studies (Blot and Fraumeni, Rencher, Sankila) were exposed to other substances, including antimony, arsenic, cadmium, chromium, copper, manganese, nickel oxide, sulphur dioxide and zinc selenite, which may themselves be potentially carcinogenic. As workers were probably exposed to one or more of these chemicals concurrently with their exposure to lead, it is not really possible to ascertain the potential contribution of any one of these substances to the lung cancer excesses observed. Furthermore, Blot and Fraumeni stated that the increased mortality found in their study was probably caused by exposure to inorganic arsenic, and not by lead or lead compounds.

In the light of these problems, it is not altogether surprising that IARC described the evidence for the carcinogenicity of lead and lead compounds to humans as "inadequate" [5].

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 73: Estimates of standardized mortality ratio for exposure to lead and lead compounds

Study	Population	Standardized mortality ratio (95% limits)
Jecklin (1956)[1]	No data	Lead levels similar in cases and controls
Blot and Fraumeni (1975)[2]	US men living near lead/zinc/ copper industries	112
	Women	110
Cooper and Gaffey (1975)[3]	US lead smelter workers	139[4]
	Battery plant workers	123[4]
Rencher et al (1977)[2]	US copper smelter workers	300
Costello (1982)[5]	US lead/zinc miners	130($p<0.05$)
Selevan et al (1985)[6]	US lead smelter workers	111($p>0.05$)[4]
Gerhardsson et al (1986)[6]	Swedish lead smelter workers	160($p>0.05$)[4]
Sweeney et al (1986)[6]	US chemical plant workers	134[4]
Sankila et al (1990)	Finnish glass factory workers	128(99-162)[7]

1 From International Agency for Research on Cancer (1972)

2 From International Agency for Research on Cancer (1980)

3 From Blot (1984)

4 Estimated from data given

5 From Goldsmith et al (1982)

6 From International Agency for Research on Cancer (1987)

7 Standardized incidence ratio

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

74. Leather industries

The studies which attempted to relate the risk of lung cancer to employment in the leather industry are detailed in Table 74. Eight standardized mortality ratios were calculated, ranging from 50 to 158, of which five were raised. Five relative risks were estimated, all of which were above 1.00, with the highest given as 4.2. Two proportional mortality ratios, of 98 and 104, were also presented, along with two proportional registration ratios, of 131 and 136.

Workers in the leather industry may be exposed to many compounds, depending on their specific occupation. The exposures of workers in leather tanning and processing industries include dusts, solvents and other chemicals used during the processes of preservation, defestation and disinfection, beamhouse process, tanning, neutralizing, retanning, bleaching, colouring or dyeing, fat liquoring, drying, pasting, and finishing. Boot and shoe manufacturers and repairers may also be exposed to dust, but the main chemical exposures come from the application of cleaners, adhesives and finishes. One study (Walker) took measurements of toluene, methyl ethyl ketone, acetone and hexane levels in the plants under study, but for all four chemicals the concentrations were found to be less than threshold levels. No data were available for other compounds, although it was noted that several others had been used by the workers. None of the other studies gave any information on the subjects' exposures.

Not surprisingly then, IARC felt that due to the design of many of the studies the evidence for a possible association between lung cancer and employment in the leather industry could not be evaluated [1]. From the evidence presented here it can be seen that if a risk does exist it does not appear to be very large.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. Goldsmith DF, Guidotti TL and Johnston DR (1982) Does occupational exposure to silica cause lung cancer? Am J Ind Med, 3, 423-440.

3. International Agency for Research on Cancer (1972) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 1, 40-50. IARC, Lyon.

4. International Agency for Research on Cancer (1980) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 23: Some metals and metallic compounds, 104 and 325-415. IARC, Lyon.

5. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 230-232. IARC, Lyon.

6. Sankila R, Karjalainen S, Pukkala E et al (1990) Cancer risk among glass factory workers: an excess of lung cancer? Br J Ind Med, 47, 815-818.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 74: Estimates of relative risk/standardized mortality ratio for employment in leather industries

Study	Population	Relative risk (95% limits)
Kennaway and Kennaway (1947)[1]	E/W tanners/leather dressers/ curriers	141*
Menck and Henderson (1949)[1]	US shoe repairers	2.33($p<0.05$)
OPCS (1978)	Male shoemakers/repairers, aged 15-64	104[2]
	Aged 65-74	98[2]
	Incidence, 1966-7	136($p<0.05$)[3]
	1968-9	131($p<0.05$)[3]
Logan (1982)	UK male shoemakers - 1931	50*
	1951	158*
	1961	154*
	1971	143*
	Married women[4] - 1951	100*
	1961	100*
Garabrant and Wegman (1984)[5]	US tannery workers	4.2($p<0.05$)
Puntoni et al (1984)[5]	Italian tannery workers	>1.00
Sweeney et al (1985)[5]	US fur tanners	>1.00($p<0.05$)
Coggon et al (1986)[5]	UK tannery workers	>1.00
Walker (1993)	US shoe factory workers	147(120-180)*

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1981)

2 Proportional mortality ratio

3 Proportional registration ratio

4 According to husband's occupation

5 From International Agency for Research on Cancer (1987)

2501208983

Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. International Agency for Research on Cancer (1981) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 25: Wood, leather and some associated industries, 201-292. IARC, Lyon.

2. International Agency for Research on Cancer (1981) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 232-237. IARC, Lyon.

3. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

4. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

5. Walker JT, Bloom TF, Stern FB et al (1993) Mortality of workers employed in shoe manufacturing. Scand J Work Environ Health, 19, 89-95.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

75. Length of menstrual cycle

Only one study presented data relevant to an investigation of the possible association between lung cancer risk in women and the length of the menstrual cycle, and details of it are given in Table 75. Relative risks of 1.6, 1.6 and 2.2 were estimated for women with a menstrual cycle of 30-33 days, 26-29 days and less than 26 days respectively.

Although the data presented in the table are suggestive of an association between lung cancer risk and length of the menstrual cycle, with only one study reporting it is not really possible to make a proper evaluation.

Table 75: Estimates of relative risk for length of menstrual cycle

Study	Population	Relative risk (95% limits)
Gao et al (1987)	Chinese women with menstrual cycle length 30-33 days	1.6(1.0-2.6)
	26-29 days	1.6(1.0-2.7)
	<26 days	2.2(1.3-3.7)

* Standardized mortality ratio

References

1. Gao Y-T, Blot WJ, Zheng W et al (1987) Lung cancer among Chinese women. Int J Cancer, 40, 604-609.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

76. Leukocyte count

Only one study was found which presented data relevant to an investigation of the possible relationship between lung cancer risk and leukocyte count, and details of it are given in Table 76. A relative risk of 1.46 was estimated for every 2000/μl increase in leukocyte count.

However, with no other studies providing data it is not possible to determine if this observed increase in risk represents a true association between lung cancer and leukocyte count.

Table 76: Estimate of relative risk for leukocyte count

Study	Population	Relative risk (95% limits)
Phillips et al (1992)	US men at high risk of heart disease/UK residents	1.46($p<0.0001$)[1]

1 Per 2000/μl increase in leukocyte count

References

1. Phillips AN, Neaton JD, Cook DG, Grimm RH and Shaper AG (1992) The leukocyte count and risk of lung cancer. Cancer, 69, 680-684.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Footnote to Table 77

1 Estimated from data given

2 Cancer sites associated with smoking (buccal cavity and pharynx, pancreas, larynx, bronchus and lung, cervix, bladder, kidney)

References

1. Anda RF, Williamson DF, Escobedo LG et al (1990) Depression and the dynamics of smoking: A national perspective. JAMA, 264, 1541-1545.

2. Covey LS, Glassman AH and Dalack GW (1991) Re: Depressed mood and development of cancer. Am J Epidemiol, 134, 324-325 (Letter).

3. Grossarth-Maticek R, Bastiaans J and Kanazir DT (1985) Psychosocial factors as strong predictors of mortality from cancer, ischaemic heart disease and stroke: The Yugoslav prospective study. J Psychosomatic Res, 29, 167-176.

4. Linkins RW and Comstock GW (1990) Depressed mood and development of cancer. Am J Epidemiol, 132, 962-972.

5. Linkins RW and Comstock GW (1991) Re: Depressed mood and development of cancer. Am J Epidemiol, 134, 325 (Letter).

6. Shekelle RB, Raynor WJ, Ostfeld AM et al (1981) Psychological depression and 17-year risk of death from cancer. Psychosomatic Med, 43, 117-125.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

77. Long-lasting hopelessness/depression

The three studies found which gave data relevant to an investigation of the possible association between lung cancer risk and depression or long-lasting hopelessness are detailed in Table 77. Six relative risks, ranging from 1.58-18.47, were estimated. Additionally, one study reported that 23.1% of bronchogenic carcinoma cases suffered from psychological depression.

Although it has been reported elsewhere that depressed subjects are less likely to quit smoking [1,2], only one of the two studies which presented data on smoking prevalence found evidence that respondents with depression were more likely to be smokers than respondents not affected in this way [5]. Depressed and non-depressed subjects in the study by Shekelle were similar to each other in their smoking habits.

Another possible explanation for the findings presented in the table is that depression may lead to immune suppression, which in turn leads to an increased rate of cancer at a variety of sites [3,4,6]. However, with so little information available it is not really possible to verify this hypothesis, or to fully evaluate the association between lung cancer and long-lasting hopelessness and depression.

Table 77: Estimates of relative risk for long-lasting hopelessness and depression

Study	Population	Relative risk (95% limits)
Shekelle et al (1981)	US men	23.1% of bronchogenic carcinoma cases had psychological depression
Grossarth-Maticek et al (1985)	Yugoslavian residents	1.58[1] ✓
Linkins and Comstock (1990)	US residents, ex-smokers	2.52(0.31-20.50)[2]
	Smokers of 1-14 cigs/day	5.80(0.71-47.19)[2]
	15-24 cigs/day	3.77(0.46-31.19)[2]
	25+ cigs/day	18.47(4.58-74.41)[2]
	Unknown smoking status	3.17(0.39-25.85)[2]

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

78. Loss of teeth

Details of the only study found which gave information relevant to an investigation of the possible association between lung cancer risk and loss of teeth are given in Table 78. It was reported that cases had lost an average of 15.9 teeth compared to only 12.6 in controls.

Notably, the authors of this study stated that "bad teeth...are, on their own, not able to cause cancer of the lungs". Until other evidence to contradict this is found, it would appear that the possible relationship between lung cancer risk and loss of teeth is in some doubt.

Table 78: Observations of number of teeth lost

Study	Population	Observations
Denoix et al (1958)	French men	Average number of teeth lost 15.9 in bronchial cancer cases and 12.6 in controls

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

79. Low forced expiratory volume

Details of the two studies which attempted to relate the risk of lung cancer to low forced expiratory volume in one second (FEV_1) are given in Table 79. Four estimates of relative risk in relation to low FEV_1 were estimated, ranging from 1.3-2.1, while a relative risk of 0.7 was estimated for those whose lung function was better than average.

The evidence presented in the table suggests a positive association between lung cancer risk and low FEV_1, but with only two studies providing data such a relationship is far from proven.

Table 79: Estimates of relative risk for low forced expiratory volume

Study	Population	Relative risk (95% limits)
Peto et al (1983)	UK men, FEV_1 better than average	0.7
	0-1 SD below average	1.5
	1-2 SD below average	1.4
	2+ SD below average	1.3
Vestbo et al (1991)	Danish men	2.1(1.3-3.4)[1]

SD = standard deviations

1 Per litre under the expected FEV_1 for height

References

1. Peto R, Speizer FE, Cochrane AL et al (1983) The relevance in adults of air-flow obstruction, but not of mucus hypersecretion, to mortality from chronic lung disease. Am Rev Respir Dis, 128, 491-500.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

2. Vestbo J, Knudsen KM and Rasmussen FV (1991) Are respiratory symptoms and chronic airflow limitation really associated with an increased risk of respiratory cancer? Int J Epidemiol, 20, 375-378.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

80. Low-level radiation

Table 80 gives details of the studies which presented data relevant to an investigation of the possible association between lung cancer risk and exposure to low-level radiation. Eight standardized mortality ratios were calculated and these ranged from 69-218, with only two being raised. Two relative risks, of 1.2 and 3.7, were also estimated. Additionally, one study estimated a risk of 25-50 per million person-rems of exposure, while another estimated a decrease in risk of 1.6% for every 10mSv of exposure.

Three studies (Darby, Mole, Petersen) gave dosimetric data based on actual measurements of the radiation exposure of respondents, while three others (Jablon and Bailar, Smith and Doll 1981, Smith and Doll 1982) attempted to estimate exposure. However, it is possible that in those studies where exposure data was not based on objective measurements, and also in the study by Kabat where no information on exposure was given at all, inaccuracies may have been introduced. Other problems to note include a possible overlap in the studies by Darby, Mole and Petersen, as these were all based on respondents from the same US plant. Finally, in the study by Kabat there was a high correlation between a history of a reproductive primary and a history of radiotherapy, and it was not possible to estimate the effect of one exposure independent of the other.

Overall, then, there is little convincing evidence of an association between lung cancer risk and exposure to low-level radiation.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 80: Estimates of standardized mortality ratio/relative risk for exposure to low-level radiation

Study	Population	Standardized mortality ratio (95% limits)
Jablon and Bailar (1980)	US residents	25-50[1]
Darby and Reissland (1981)	US nuclear plant workers	75[2]
Smith and Doll (1981)	UK male radiologists entering study before 1921	218(p<0.05)
	Entering study 1921-1954	97
Smith and Doll (1982)	UK ankylosing spondylitis patients	142(p<0.001)
Mole (1987)	UK radiation workers	69(p<0.01)
	UK radiation workers	87(p<0.05)
	US radiation workers	78
	US radiation workers	75(p<0.05)
Petersen et al (1990)	US radiation workers	-1.6%(<0-5.6%)[3]
Kabat (1993)	US male radiotherapy patients	1.2(0.2-6.4)*
	Females	3.7(1.2-10.9)*

* Relative risk

1 Risk per million person-rems

2 Tumours of respiratory system

3 Estimated increase in relative risk per 10 mSv

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Darby SC and Reissland JA (1981) Low levels of ionizing radiation and cancer - are we underestimating the risk? J R Statist Soc, 144, 298-331.

2. Jablon S and Bailar JC (1980) The contribution of ionizing radiation to cancer mortality in the United States. Prev Med, 9, 219-226.

3. Kabat GC (1993) Previous cancer and radiotherapy as risk factors for lung cancer in lifetime nonsmokers. Cancer Causes and Control, 4, 489-495.

4. Mole RH (1987) Radiation, cancer risk, and the new dosimetry. The Lancet, Dec 12, 1403-1404.

5. Petersen GR, Gilbert ES, Buchanan JA and Stevens RG (1990) A case-cohort study of lung cancer, ionizing radiation, and tobacco smoking among males at the Hanford site. Health Phys, 58, 3-11.

6. Smith PG and Doll R (1981) Mortality from cancer and all causes among British radiologists. Brit J Radiol, 54, 187-94.

7. Smith PG and Doll R (1982) Mortality among patients with ankylosing spondylitis after a single treatment course with x rays. Brit Med J, 284, 449-60.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

81. Lung scars

Four studies attempted to investigate the possible relationship between lung cancer risk and lung scarring, and details of these are given in Table 81. A total of 36 cases of lung cancer associated with lung scarring were reported.

However, from the data given it was not possible to calculate any estimates of risk of lung cancer in individuals with lung scarring, and so it is difficult to properly evaluate the evidence.

Table 81: Observations on lung cancer associated with lung scarring

Study	Population	Observations
Carroll (1962)	UK residents	13 of 109 lung cancers associated with lung scarring
Raeburn and Spencer (1957)	UK residents	15 lung cancers associated with lung scarring reported
Strauss et al (1963)	US/UK/French/German residents	11 lung cancers associated with embedded foreign bodies reported
Yokoo and Suckow (1961)	US residents	7 out of 41 lung cancers associated with lung scarring

References

1. Carroll R (1962) The influence of lung scars on primary lung cancer. J Path Bacteriol, 83, 293-297.

2. Raeburn C and Spencer J (1957) Lung scar cancers. Brit J Tuberc, 51, 237-245.

3. Strauss FH, Dordal E and Kappas A (1963) The problems of pulmonary scar tumors: A case report and brief review. Arch Path, 76, 693-699.

4. Yokoo H and Suckow EE (1961) Peripheral lung cancers arising in scars. Cancer, 14, 1205-1215.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

82. Man-made mineral fibres

Details of the studies which gave information relevant to an investigation of the possible association between lung cancer risk and exposure to man-made mineral fibres are given in Table 82. Ten standardized mortality ratios were calculated and these ranged from 68-200, with nine being raised. Additionally, two studies reported standardized incidence ratios, of 74 and 91, while another found that four cases and no controls had worked in a glasswool plant.

Four studies (Enterline, Gustavsson, Saracci, Shannon) had made objective measurements of the level of man-made mineral fibres respondents were exposed to, while the study by Simonato had attempted to quantify exposure by length of employment. However, no information on the workers' exposure was available from the other studies, which may therefore contain inaccuracies. Furthermore, it was stated that in the study by Engholm respondents were exposed to asbestos in addition to man-made mineral fibres, although, surprisingly, this study reported a decrease in lung cancer incidence.

While IARC felt that there was "limited" evidence of the carcinogenicity of rock/slagwool to humans, that for glasswool and glass filaments was classified as "inadequate" [2]. A review by Lippmann described the risk of lung cancer following exposure to man-made mineral fibres as "virtually nil" [3].

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 82: Estimates of standardized mortality ratio for exposure to man-made mineral fibres

Study	Population	Standardized mortality ratio (95% limits)
Bayliss et al (1976)[1]	US glasswool plant workers	Four cases and no controls worked in plant
Morgan et al (1984)[1]	US fibrous glass plant workers	136[3]
Saracci et al (1984)[2]	7 European countries	200
Moulin et al (1986)[1]	French glass-fibre production workers	74(24-172)[4]
Engholm et al (1987)[1]	Swedish construction workers	91(83-100)[4]
Enterline et al (1987)[1]	US small diameter fibre workers	133[3]
	Glass filament workers	95[3]
	Rock/slagwool workers	148[3]
Shannon et al (1987)[1]	Canadian wool plant workers	199($p<0.05$)
Simonato et al (1987)[1]	European glasswool plant workers	127($p<0.05$)
	Glass filament workers	120
	Rock/slagwool workers	124(98-154)
Gustavsson et al (1992)	Swedish factory workers	68(37-113)

1 From International Agency for Research on Cancer (1988)

2 From Lippmann (1992)

4 Standardized incidence ratio

3 Tumours of respiratory system

2501208997

Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Gustavsson P, Plato N, Axelson O et al (1992) Lung cancer risk among workers exposed to man-made mineral fibers (MMMF) in the Swedish prefabricated house industry. Am J Ind Med, 21, 825-834.

2. International Agency for Research on Cancer (1988) Monographs on the evaluation of carcinogenic risks to humans. Volume 43: Man-made mineral fibres and radon, 39-171. IARC, Lyon.

3. Lippmann M (1992) Asbestos and other mineral fibers. In: Environmental Toxicants: Human Exposures and Their Health Effects. (Incomplete reference)

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

83. Marine diesel fuels

Table 83 gives details of the only study which attempted to relate the risk of lung cancer to exposure to marine diesel fuels. A relative risk of 1.6 was estimated.

It was noted that no attempt was made in the study to separate the effects of exposure to the combustion products of marine diesel fuels from those of exposure to the liquid itself, and thus it is possible that the observed excess of lung cancers could have been caused by a constituent of the fuel. In the light of this, and with only one study reporting, IARC justifiably classified the evidence for the carcinogenicity of marine diesel fuels to humans as "inadequate" [1].

Table 83: Estimate of relative risk for marine diesel fuels

Study	Population	Relative risk (95% limits)
Siemiatycki et al (1987)[1]	Canadian men	1.6(1.0-2.6)

1 From International Agency for Research on Cancer (1989)

References

1. International Agency for Research on Cancer (1989) Monographs on the evaluation of carcinogenic risks to humans. Volume 45: Occupational exposures in petroleum refining; crude oil and major petroleum fuels, 219-237. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

84. Maternal age at birth

Only one study attempted to relate the risk of lung cancer to the age of the mother at the time of the subject's birth, and details of it are given in Table 84. It was reported that the average maternal age for cases was 28.629 years, compared to 27.420 years for controls.

With only one study reporting it is not possible to determine if there is an effect of maternal age at birth on subsequent lung cancer risk in the offspring or whether the reported association arose simply by chance.

Table 84: Observations for maternal age at birth

Study	Population	Observations
Abelin and Tokuhata (1965)	US residents	Average age for cases 28.629 years compared to 27.420 for controls

References

1. Abelin T and Tokuhata GK (1965) Maternal age at birth and susceptibility to lung cancer. Lancet, Nov 25, 1121-1123.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 86: Estimates of relative risk/standardized mortality ratio for exposure to mercury and mercury compounds

Study	Population	Relative risk (95% limits)
Milham (1976)[1]	US dentists	105(88-125)[2]
Costello (1982)[3]	US mercury miners	355(p<0.05)*
Cragle (1984)[4]	US workers in nuclear weapons factory	134(100-180)*
Buiatti et al (1985)[4]	Italian residents	6 cases and no controls ever employed as hat makers (p<0.01)
Walrath et al (1985)[4]	US dentists	1.00
Tamashiro et al (1986)[1]	Japanese fishermen and families	1.52(0.79-2.65)
Gallagher et al (1989)[4]	Canadian dentists	1.00
Barregard et al (1990)[4]	Swedish chloralkali workers	180(90-300)[5]
Amandus and Costello (1991)[4]	US mercury miners - silicotics	1400(289-4100)*
	Nonsilicotics	266(115-524)*
Ellingsen et al (1991)[4]	Norwegian chloralkali workers	166(100-260)[5]
Siemiatycki (1991)[4]	Canadian residents	4.0(1.2-13.0)

* Standardized mortality ratio

1 From International Agency for Reseach on Cancer (1993)

2 Proportional mortality ratio

3 From Goldsmith et al (1982)

4 From Boffetta et al (1993)

5 Standardized incidence ratio

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Boffetta P, Merler E and Vainio H (1993) Carcinogenicity of mercury and mercury compounds. Scand J Work Environ Health, 19, 1-7.

2. Goldsmith DF, Guidotti TL and Johnston DR (1982) Does occupational exposure to silica cause lung cancer? Am J Ind Med, 3, 423-440.

3. International Agency for Research on Cancer (1993) Monographs on the evaluation of carcinogenic risks to humans. Volume 58: Beryllium, cadmium, mercury, and exposures in the glass manufacturing industry, 239-345. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

85. Meat consumption

Table 85 gives details of the three studies found which presented data relevant to an investigation of the possible association between lung cancer risk and frequent meat consumption. Three relative risks were estimated, ranging from 1.29-1.53.

Although the evidence presented in the table is indicative of an association between lung cancer risk and the frequent consumption of meat, data from further studies is needed before this relationship can be confirmed.

Table 85: Estimates of relative risk for frequent meat consumption

Study	Population	Relative risk
Hirayama (1974)	Japanese men	1.53
Fraser et al (1991)	US Seventh-day Adventists	1.31(0.52-3.28)
Alavanja et al (1993)	US women	1.29

References

1. Alavanja MCR, Brown CC, Swanson C and Brownson RC (1993) Saturated fat intake and lung cancer risk among nonsmoking women in Missouri. JNCI, 85, 1906-1916.

2. Fraser GE, Beeson WL and Phillips RL (1991) Diet and lung cancer in California Seventh-day Adventists. Am J Epidemiol, 133, 683-93.

3. Hirayama T (1974) Prospective studies on cancer epidemiology based on census population in Japan. Proc 11th International Cancer Congress, 3, 26-35.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

86. Mercury and mercury compounds

Table 86 gives details of the 11 studies which considered lung cancer risk in relation to exposure to mercury and mercury compounds. Four relative risks were estimated, ranging from 1.00-4.00, and two were above 1.00. Four standardized mortality ratios, of between 134-1400, were calculated, along with two standardized incidence ratios, of 166 and 180, and one proportional mortality ratio of 105. In addition, one study found that six cases and no controls had ever been employed as hat makers.

Only three studies (Barregard, Cragle, Ellingsen) took objective measurements of exposure to mercury, by recording mercury levels in the subjects' urine. Thus, in the other studies, inaccuracies due to exposure misclassification could have been introduced. Additionally, some of the studies reported that subjects had been exposed to various other substances which may themselves be potential carcinogens, including asbestos and static magnetic fields (Barregard), arsenic and other chemicals (Buiatti), radon (Amandus and Costello) and silica (Amandus and Costello, Costello). It is therefore possible that the observed associations in these studies may have been caused by exposure to a substance other than mercury. In addition, Cragle et al suggested that the excess of lung cancer observed in their study may be due to lifestyle factors or some factor other than mercury.

Thus, when evaluating the evidence for the carcinogenicity of mercury and mercury compounds to humans, IARC gave it a classification of "inadequate" [3].

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

87. Metal workers

Only four studies were found which attempted to relate the risk of lung cancer to employment in the metal working trades, and these are detailed in Table 87. Four standardized mortality ratios were calculated, ranging from 115-170. Six proportional mortality ratios were presented, and these lay in the range 105-113. Six proportional registration ratios, of between 117 and 134, were also given. Two studies estimated relative risks, of 1.26 and 1.3.

Previous studies of metal workers have observed associations between cancer of various sites and a number of compounds, including nitrosable amines and nitrites from cutting oils, water based cutting oils, straight oils, polycyclic aromatic hydrocarbons suspended in oil mists, chlorinated hydrocarbon solvents used for degreasing metal parts, and metal dust resulting from machining and grinding operations [1]. However, as only the study by Acquavella attempted to classify the substances the subjects were exposed to it is not really possible to identify the potential carcinogenicity of any one compound.

The data presented in the table is suggestive of an increased risk of lung cancer among metal workers, but due to a lack of information the agent or agents responsible for this increase cannot be identified. It should also be remembered that the evidence is based on results from only four studies.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 87: Estimates of relative risk/standardized mortality ratio for metal workers

Study	Population	Relative risk (95% limits)
OPCS (1978)	E/W male sheet metal workers, aged 15-64	110[1]
	Aged 65-74	106[1]
	Incidence, 1966-7	128(p<0.01)[2]
	1968-9	122(p<0.01)[2]
	Male metal plate workers/riveters, aged 15-64	113[1]
	Aged 65-74	105[1]
	Incidence, 1966-7	131(p<0.01)[2]
	1968-9	134(p<0.01)[2]
	Male other metal making/working/jewellery electrical production process workers, aged 15-64	110(p<0.05)[1]
	Aged 65-74	108[1]
	Incidence, 1966-7	117(p<0.01)[2]
	1968-9	117(p<0.01)[2]
OPCS (1986)	UK male sheet metal workers/platers/ shipwrights/riveters	170(p<0.01)*
	Married women[3]	142(p<0.01)*
	Male metal working production fitters and fitter/machinists	115(p<0.01)*
	Married women[3]	124(p<0.01)*
Acquavella et al (1993)	Metal components workers	1.3(0.9-1.8)
Keller and Howe (1993)	US construction metal workers	1.26(0.67-2.35)

* Standardized mortality ratio

1 Proportional mortality ratio

2 Proportional registration ratio

3 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Acquavella J, Leet T and Johnson G (1993) Occupational experience and mortality among a cohort of metal components manufacturing workers. Epidemiol, 4, 428-434.

2. Keller JE and Howe HL (1993) Cancer in Illinois construction workers: A study. Am J Ind Med, 24, 223-230.

3. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

4. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

88. Metronidazole

Only two studies were found which presented data relevant to an investigation of the possible association between lung cancer risk and exposure to metronidazole, and details of these are given in Table 88. Two standardized mortality ratios, of 77 and 667, were calculated.

With so few studies reporting, it is difficult to evaluate the carcinogenicity of metronidazole. Furthermore, additional follow-up and analysis of the data from the study by Beard suggested that the excess of lung cancer observed could be explained entirely by confounding with smoking. Thus, IARC concluded there was "inadequate" evidence for the carcinogenicity of metronidazole to humans [1].

Table 88: Estimates of standardized mortality ratio for exposure to metronidazole

Study	Population	Standardized mortality ratio
Beard et al (1979)[1]	Female trichomoniasis patients	667[2]
Friedman (1980)[1]	Female trichomoniasis patients	77[2]

1 From International Agency for Reseach on Cancer (1987)
2 Estimated from data given

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 250-252. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

89. Mineral oils

Details of the 13 studies which attempted to relate the risk of lung cancer to exposure to mineral oils are given in Table 89. Eight standardized mortality ratios were calculated, ranging from 56-149, of which five were raised. Two relative risks were also estimated, at 1.73 and 3.76. Additionally, one study calculated an annual mortality rate of 1.07 per 1000, while one reported excesses of lung cancer among workers in occupations in which exposure to mineral oils was likely. Finally, one study observed 2.9% of deaths due to lung cancer among the exposed group compared to 3.2% in the control group.

Exposure to mineral oils occurs in a variety of industries, and it is highly likely that workers are also exposed to other substances which may themselves be potentially carcinogenic. For example, printing pressmen are exposed to oil mist containing carbon blacks, pitch, other pigments and additives, while the sandblasters in the study by Puntoni were noted to have been exposed to many solvents, naphtha, and silica sand. In addition, insufficient information was given by the studies to allow an assessment of the class of mineral oil, which exists in many forms, workers were exposed to. Therefore, it is difficult to determine the carcinogenic potential of any one substance.

Despite these drawbacks, IARC [2,3] felt that although there was "sufficient" evidence for the carcinogenicity to humans of untreated and mildly treated mineral oils, that for highly refined oils was "inadequate". However, these evaluations were based mainly on studies of skin cancer.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 89: Estimates of standardized mortality ratio/relative risk for exposure to mineral oils

Study	Population	Standardized mortality ratio
Ely et al (1970)[1]	US metalworkers	2.9% deaths from resp. system cancer against 3.2% in controls
Goldstein et al (1970)[1]	US newspaper plant workers	1.07[2]
Greenberg (1972)[1]	UK newspaper printers	133($p<0.01$)[3]
Moss et al (1972)[1]/Moss (1973)[1]	UK newspaper workers	134($p<0.01$)[3]
Decoufle (1976)[1]	US metalworkers	99[3,4]
Menck and Henderson (1976)[1]	US newspaper printers	98
Milham (1976)[1]	US mechanics/repairmen, tool/die makers/setters, oilers/greaser, pressmen/ plate printers	Excesses of lung cancer reported
Lloyd et al (1977)[1]	US newspaper pressmen	112[3]
OPCS (1978)[1]	UK machine-tool setters/ setter operators, motor mechanics/auto engineers, fitters, machine erectors	>100
Puntoni et al (1979)[5]	Italian sandblasters	3.76($p<0.05$)*
Paganini-Hill et al (1980)[1]	US newspaper pressmen	149[3]
Jarvholm et al (1981)[1]	Swedish metalworkers	56[3]
Zappa et al (1993)	Italian weavers	1.73(1.1-2.7)*

* Relative risk

1 From International Agency for Research on Cancer (1984)

2 Per 1000 per year

3 Estimated from data given

4 Tumours of respiratory system

5 From Goldsmith et al (1982)

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

91. Mustard gas

Details of the four studies which investigated the possible association between lung cancer risk and exposure to mustard gas are given in Table 91. Four standardized mortality ratios were calculated, ranging from 147-3667.

Only the study by Wada contained any information on the level of exposure to mustard gas, giving objective measurements of workplace concentrations. Thus, there may have been inaccuracies in the other studies due to misclassification of exposure. Additionally, workers in the study by Weiss and Weiss were also exposed to nitrogen mustard, bromoacetone, phosgene, chloropicrine and organic arsenicals. It is possible that the increased lung cancer risk observed in this study may have been caused by one or more of these substances. Interestingly, Case and Lea concluded that the increased risk of lung cancer observed in their study could not be attributed to mustard gas poisoning but was in fact associated in some way with chronic bronchitis.

Despite this, IARC felt there was "sufficient" evidence for the carcinogenicity of mustard gas to humans [3], and a review by Blot described it as a "known" occupational lung carcinogen [1].

Table 91: Estimates of standardized mortality ratio for exposure to mustard gas

Study	Population	Standardized mortality ratio
Case and Lea (1955)[1]	War pensioners	207[2]
Beebe (1960)[1]	American soldiers	147[3]
Wada et al (1968)[4]	Japanese mustard gas workers	3667[2]
Weiss and Weiss (1975)[1]	German mustard gas workers	280[2,5]

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Footnote to Table 91

1 From International Agency for Research on Cancer (1975)

2 Estimated from data given

3 Tumours of respiratory system

4 From Blot (1984)

5 Bronchial carcinoma

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. International Agency for Research on Cancer (1975) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 9: Some aziridines, N-, S- and O-mustards and selenium, 181-192. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 322-326. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

Table 90: Relationship between lung cancer and month of birth

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Dijkstra (1963):												
O	23	36	50	29	19	22	21	20	26	28	29	27
E	28.67	27.81	29.68	28.04	27.71	25.58	26.45	27.65	27.52	27.37	26.03	27.51
Jones (1963)[1]:												
O	--14--		--17--		--21--		--15--		--15--		--13--	
Loxton (1963)[1]:												
O	--116--		--113--		--115--		--144--		--138--		--115--	
Baas and Strackee (1964)[1]:												
O	124	113	132	118	87	103	106	114	123	118	106	102
Davies (1964)[1]:												
O	170	169	173	173	162	158	158	189	185	165	172	168
De Sauvage Nolting (1964):												
O	--- Higher than overall rate ---				--- Lower than overall rate ---				-- Not reported --		As Jan-Apr	
MacSween and Miller (1964):												
Ca	22	9	9	22	19	19	20	21	14	13	18	14
Co	18	16	24	17	20	19	13	16	11	15	12	19
Palmer (1964):												
O	3352	3193	3426	3230	3360	3226	3280	3363	3267	3338	3223	3392
Van der Wal et al (1964)[1]:												
O	10	15	17	9	9	10	13	18	15	13	11	10

Ca = Cases; Co = Controls; E = Expected; O = Observed

1 From Allan (1964)

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Allan TM (1964) Lung cancer and month of birth. The Lancet, Feb 22, 439-440.

2. Anon (1963) Lung cancer and month of birth. The Lancet, Dec 7, 1210-1211.

3. Cochrane AL and Palmer JW (1964) Lung cancer and date of birth. The Lancet, Dec 5, 1246 (Letter).

4. Davies J (Incomplete reference)

5. De Sauvage Nolting WJJ (1964) Lung cancer and month of birth. The Lancet, Sep 5, 531 (Letter).

6. Dijkstra BKS (1963) Origin of carcinoma of the bronchus. JNCI, 31, 511-519.

7. MacSween RNM and Miller SEP (1964) Lung cancer and month of birth. The Lancet, Apr 4, 767-768 (Letter).

8. Palmer JW (1964) Lung cancer and month of birth. The Lancet, Nov 7, 1013 (Letter).

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

References

1. Goldsmith DF, Guidotti TL and Johnston DR (1982) Does occupational exposure to silica cause lung cancer? Am J Ind Med, 3, 423-440.

2. International Agency for Research on Cancer (1984) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 33: Polynuclear aromatic compounds, part 2, carbon blacks, mineral oils and some nitroarenes, 87-168. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 252-254. IARC, Lyon.

4. Zappa M, Paci E, Costantini AS and Kriebel D (1993) Lung cancer among textile workers in the Prato area of Italy. Scand J Work Environ Health, 19, 16-20.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000

90. Month of birth

In 1963, Dijkstra first suggested that there may be a higher risk of lung cancer in people born in late winter, following the observation of an excess of lung cancer deaths in patients born in March in a series of 330 deaths occurring between 1950 and 1960 in the Netherlands. A further 8 studies were found which also attempted to relate lung cancer risk and month of birth in this way, and the results of these 8 studies are given in Table 90. From the table it can be seen that in three of the studies there does appear to be an excess of births in March, and an excess of births in the winter months is also seen in one other study. However, due to the failure of most of the studies to give any estimates of the number of births which would be expected in any one month it is difficult to assess the significance of any differences in the actual numbers of births observed.

It is hardly likely that month of birth in itself is a risk factor for lung cancer, and so it is more probably a marker for some other factor. One suggestion is that the excess of lung cancer deaths in those born in late winter can be explained by the absence of vitamin A at birth, causing irreversible metaplasia in the lungs. This leaves the tissue unable to cope with the normal stimuli of life and predisposes the individual to various pulmonary diseases, including cancer [2,6]. Although this may be a possible explanation, the evidence for an association between month of birth and lung cancer as presented here is hardly compelling.

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Source: https://www.industrydocuments.ucsf.edu/docs/mhhl0000